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The Management of Postpartum Hemorrhage

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A PHYSICIAN who is responsible for even one obstetric patient has the threat of a postpartum hemorrhage hanging over him. He may avoid the problem of diabetes in pregnancy by simply referring diabetic pregnant patients elsewhere for care. Similarly, the problem of heart disease in pregnancy can be avoided. But there is no way he can avoid the problem of postpartum hemorrhage, for there is no warning sign by which it can be anticipated. Postpartum hemorrhage remains, therefore, a topic about which all of us must think and for which each of us must be prepared.

The recent interest which has been aroused by the phenomenon of hypofibrinogenemia has served the very important function of redirecting attention to some of the mechanisms which are involved in this dread complication. This same revival of interest, however, and this same body of literature has had the disadvantage of making a statistically uncommon cause of hemorrhage appear to be far more frequent that it actually is. Upon careful review of the literature on hypofibrinogenemia it can be observed that some patients have been reported from more than one institution, and that in many of the case reports there is the *presumption* of a decline in fibrinogen rather than *proof* of fibrinogen depletion. Of this mass of articles one might safely say that seldom has so much medical literature been owed to so few patients based on so small a number of laboratory determinations. While the problem is an important one it is not a frequent one, and it would be well to consider the subject of postpartum hemorrhage with that in mind.

Table 1 indicates the frequency of obstetric hemorrhage at MacDonald House, Cleveland, calculated per 1,000 live births. As MacDonald House is a teaching hospital, there may be an increased frequency of complicated obstetric problems referred to the obstetrical service, but in general this is probably an accurate reflection of the experience of many hospitals. It can be seen from this table that of every 100 women who delivered at term, about 15 had some form of obstetrically-related bleeding. But it can also be seen from this table that even in an institution receiving such desperate referrals, the problem of proven hypofibrinogenemia is far less frequent than is the problem of postpartum hemorrhage stemming from other causes.

Therefore, considering these topics not from the point of view of the amount of attention they have been receiving recently, but from the point of view of the frequency of occurrence in the delivery room, it might be well to start by asking ourselves the question: What makes the postpartum patient stop bleeding? In normal circumstances, why does not a woman become completely exsanguinated in the first few minutes following the delivery of the placenta? In an effort to approach the answers to these questions, some time ago a series of careful measurements of postpartum blood loss was carried out. During the period of the study the various

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TABLE 1.—Causes Obstetric Hemorrhage (Stated in Number of Cases per 1,000 Live Births) Observed on Service of Department of Obstetrics and Gynecology, Western Reserve University School of Medicine

Abortion	95.3
Therapeutic abortion	2.4
Ectopic pregnancy	8.3
Abruptio placenta	
With stillbirth	5.1
Live born	19.6
Placenta praevia	5.3
Low implantation	2.8
Postpartum hemorrhage	13.2
Rupture uterus	0.5
Placenta accreta	0.5
Hypofibrinogenemia	1.2

factors which it was felt might influence the duration and amount of postpartum bleeding were deliberately altered.

Initially the patients were given dicumarol during labor and on into the puerperium, and although the prothrombin level of the treated patients was held considerably below that of the control group, there was no increase in the amount of postpartum bleeding. Similarly, a group of women were given heparin, which increased the clotting time during the immediate puerperium to between 20 and 30 minutes, and again when compared with the control group of patients there was no increased loss of blood from the uterus. During the time that these studies were being carried out, the opportunity arose to deliver a patient who had a platelet count of about 64,000 per at the time of delivery. She had bleeding into the renal pelvis and rather constant hematuria. There was bleeding from the gums and multiple ecchymosis, but there was no increased loss of blood from the interior of the uterus. Similarly, in cases reported in the literature in which delivery was done in the face of thrombocytopenia, one finds a record of hemorrhage from abdominal incisions or into episiotomy wounds, but not the frequent occurrence of postpartum bleeding from within the uterus proper.

In a negative sense, it would appear, therefore, that the answer to the question as to what stops postpartum bleeding does not lie in the prothrombin level of the blood (actually the postpartum patient normally has a prothrombin value of between 110 and 125 per cent) nor in the clotting time of circulating blood, nor in the platelet count. One must look elsewhere than in these fractions of the clotting mechanism to find the normal control of postpartum blood loss.

As these measurements were continued and the increased loss of blood was related to the various possible factors observable in the course of the patient's pregnancy, labor and delivery, a variety of situations were encountered which would increase the loss of blood and even on occasion put it over 500 cc. and into the range of postpartum hemorrhage. The depth of anesthesia and the agent used had such an influence; so also did retained secundines; increasing parity and overdistention of the uterus as with twins had a significant association with increased amounts of postpartum bleeding.

All these factors, of course, simply contribute to diminishing the efficiency of the postpartum uterine contraction. The study served to reemphasize that, for the average patient in normal circumstances, the control of the amount of blood lost from the interior of the uterus is principally based on the efficiency of the uterine contraction. Actually, the open sinuses of the placental bed represent a tremendous defect in the integrity of the patient's vascular system. If a surface area were dissected off the anterior wall of the vena cava, proportionate to the defect that is represented by the open maternal sinuses in the placental bed, the patient would be expected to exsanguinate quite quickly. Yet this same sized defect does exist in each parturient at the time the placenta is delivered, despite which the loss of blood stays within reasonable bounds. In usual circumstances, obviously, this large defect is closed not by the patient's clotting mechanism, but by the ligature effect of the myometrial muscle bundles. The contraction of the myometrium associated with and following the expulsion of the placenta serves to close the maternal sinuses and to prevent excessive blood loss. If this ligature is effective, then administering dicumarol or heparin will not measurably alter the bleeding. Stating this principle not from the point of view of etiology, but from the point of view of therapy, we could say that the contracted uterus does not bleed, and that therapeutic efforts should be initially directed toward getting the uterus effectively contracted. Therefore, a rational approach to the problem of the control of postpartum bleeding would be a review of those factors which commonly can interfere with the effectiveness of the uterine contraction.

Under the heading of the systemic causes of ineffective myometrial contraction, we should note:

A. General anesthesia. Faced with a tetanically contracting uterus and the threat of uterine rupture, a physician would instantly think of ether, because of its known paralyzing effect on the uterus. Yet it is remarkable how often it is forgotten that a bleeding patient who has been under a general anesthesia must be reawakened as rapidly as possible. When the objective is to achieve efficient uterine contraction, the presence of the most effective known uterine relaxer in the patient's system is obviously an enemy.

B. Shock. The patient in surgical shock has an ineffective contraction of the biceps and it is absurd to expect her to have effective contraction of the

uterus. A woman in shock has poor uterine contractility, and poor uterine contractility spells increased uterine bleeding.

C. Anemia and debility. The expression "blood loss breeds the loss of blood" stems from the diminished effectiveness of uterine contraction in this group of patients.

Those causes which are uterine would include the overdistention associated with twins or with a mole; intramural fibroids; and lacerations of the uterus sustained during delivery itself. Those local factors which are intrauterine are represented overwhelmingly by retained secundines and the presence of intrauterine "clots." The word "clot" is placed in quotation marks because, of course, postpartum blood by itself cannot clot. It has no clotting mechanism, and even the addition of calcium or fibrinogen or of thromboplastin will not result in its clotting. This nonclotting blood from the puerperal uterus can, however, be caused to coagulate by seeding it with small amounts of venous blood. The resultant clot is jelly-like, noncontractual, and ineffective in so far as hemostasis is concerned. Indeed such a clot, by holding open the placental bed, can increase the amount of blood a patient loses.

These factors—each of them contributing to an increase in the amount of blood that the postpartum patient loses—are stressed here not because of their newness, nor to deny the importance of the hypofibrinogenemia state, but rather to reemphasize the fact that a successful and efficient contraction of the uterus is one of the essentials in puerperal hemostasis.

It must be pointed out, however, that this uterine contraction never provides an entirely successful ligature. If it did, there would be no postpartum blood from the uterus at all; if it did, the presence of hypofibrinogenemia might not result in uterine hemorrhage.

Hypofibrinogenemia can be caused by three obstetric conditions: (1) The retention of a dead fetus in a missed abortion; (2) amniotic fluid embolus; (3) abruptio placenta. When a depletion of the fibrinogen level takes place in association with any of these conditions, it can lead to massive loss of blood through that portion of the placental bed which is not "ligated" by the uterine contraction.

With respect to the intrauterine retention of a dead fetus, hypofibrinogenemia will not develop if fetal death has taken place before the fourth month of gestation, and does not appear until at least five weeks after fetal death. If the patient fulfills these criteria of having achieved more than four months of pregnancy and of carrying a dead fetus for more than five weeks, fibrinogen determinations should be made at regular intervals. It is well to remember that a depletion of the fibrinogen in the circulating blood does not occur in all such cases. Pritchard and Ratnoff performed such serial fibrinogen determinations on 31 women after fetal death, and in 23 of them found no evidence of a critical depletion of fibrinogen. Any therapeutic interruption of the pregnancy, however, should be done only with accurate knowledge of the fibrinogen level and only when adequate fibrinogen for replacement is present.

As to amniotic fluid embolus, it is well to remember that the patient is in shock. Replacement of the fibrinogen and evacuation of the uterus will not by themselves completely stem the uterine hemorrhage unless the shock is treated simultaneously with oxygen, transfusions and vasopressor drugs.

With respect to abruptio placenta, it should also be noted that not all patients with either partial or complete abruptio have depletion of circulating fibrinogen. Diagnosis in the form of a determination of fibrinogen levels is imperative before treatment can be intelligent or effective.

A method for rapid determination of fibrinogen levels stems from a suggestion made by Page. One centimeter of the patient's venous blood when added to two drops of reconstituted topical thrombin will immediately clot regardless of the fibrinogen level. However, within one or two minutes, in the presence of hypofibrinogenemia, this clot will contract and extrude its red cells; whereas, with adequate levels of fibrinogen this clot will remain intact. This determination gives a sufficiently rapid and sufficiently accurate indication of the fibrinogen levels that it should be carried out promptly on all patients with postpartum uterine bleeding. Fibrinogen therapy should not be carried out unless this test indicates necessity for it, and should be persisted in until the test indicates the reestablishment of normal levels.

With these principles in mind, it would be well to review the steps that should be taken in the management of a patient with postpartum hemorrhage:

A. Exploration of the lower uterine tract. In one of the most dramatic cases of postpartum hemorrhage observed by the author in the past 18 months, the patient was transferred to MacDonald House from a nearby community with presumed hypofibrinogenemia. Upon careful examination, a paracervical laceration in the left fornix was noted. The romance of the possible diagnosis of hypofibrinogenemia had resulted in overlooking a source of bleeding that was cured with two sutures.

B. Exploration of the interior of the uterus. It is imperative to know that the uterus is empty of any large placental portions and that it is not lacerated. If a physician is to be slow and reluctant to put an examining hand into the interior of the uterus, then his patients are not truly benefiting from the age of the sulfonamides and antibiotics.

C. Uterine evacuation. Both by the exploring hand, as well as by curettage—either with a gauze curette of a sponge over the fingers or with a large sized metal curette and placental forceps—the physician must make sure that the uterus is properly evacuated.

D. Holding the uterus. With a hand in the vagina and a hand on the abdominal wall, the uterus can be compressed, temporarily abating the flood of blood. Such compression not only replaces the contractile force of the myometrial muscle bundles temporarily, but also provides manual stimulation to irritate the uterus into its own contraction.

E. Oxygenation of the patient. A patient who has been under general anesthesia must be "flushed out" and awakened as rapidly as possible. A patient who has been under regional block will likewise benefit from the administration of oxygen at this point.

F. The prompt placement of an intravenous needle. Initially infusion of 5 per cent glucose solution can be started and it is well to add 1 cc. of Pitocin (oxytocin injection U.S.P.) to the first bottle of fluid. The presence of a large needle in the vein beforehand (to avoid difficulty in placing it in event of peripheral venous collapse), the administration of the fluid itself and the continuous intravenous administration of Pitocin are all of imperative importance at this moment.

Such a therapeutic approach represents a balanced attack on the problem of postpartum hemorrhage, which is based on the fundamental principles involved and the frequency with which the various entities are encountered. Three additional comments should perhaps be made:

1. What is the place of intrauterine packing in a regimen such as this? Probably, in accordance with these principles, the answer is that it has no place at all. On the one hand, the manual compression of the uterus can provide a greater occlusive force to the placental sinuses than can gauze, and on the other hand the presence of the packing within the uterus works against the fundamental principle, herein advocated, that the empty contracted uterus will not bleed. Probably in those circumstances in which gauze packing is actually placed within the uterine cavity proper instead of in the lower uterine segment, it serves on the one hand only to hold the placental sinuses open, and on the other hand, like the wick in a kerosene lamp, serves as an easy route to conduct the blood to the outside.

2. It is well to bear in mind with regard to oxytocic medications that both the alkaloids of ergot and the extracts of the posterior pituitary are nitrogenous products which, in solution, gradually lose their potency. Thus, ergotrate bears an expiration date, although the fact is overlooked by many hospital purchasing agents. It should be icebox-stored, although most hospital pharmacists store it on the open shelves. It should be transferred to the delivery room in small amounts and the ampules used in sequence, although many floor supervisors persist in ordering an estimated week's supply and letting it languish in the delivery room several days while it loses strength. A certain amount of "policing" of the logistics of the oxytocic medications, getting them from the manufacturer to the patient in short order and good condition will reduce the number of cases of excessive bleeding after delivery.

3. Blood replacement remains imperative. It has been truly said: "The hemorrhaging patient dies from the loss of one red blood cell." The body mechanisms can adjust to progressive hemorrhage up to a certain point. At that point the homeostatic adjustments to hemorrhage are defeated. Unfortunately, the physician does not know which red blood cell may represent the turning point. All of them must be replaced, and replaced promptly, to save a patient with severe bleeding.

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